DASA 2461

THE ACUTE EFFECTS OF AIR BLAST ON PULMONARY FUNCTION IN DOGS AND SHEEP

Technical Progress Report
on
Contract No. DA-49-146-XZ-372

Edward G. Damon, John T. Yelverton, Ulrich C. Luft,
Kabby Mitchell, Jr., and Robert K. Jones

March 1970

THIS WORK, A PHASE OF INVESTIGATIONS DEALING WITH THE BIOLOGICAL EFFECTS OF BLAST FROM BOMBS, WAS SUPPORTED BY THE DEFENSE ATOMIC SUPPORT AGENCY OF THE DEPARTMENT OF DEFENSE.

PREPARING AGENCY
Lovelace Foundation for Medical Education and Research
Albuquerque, New Mexico

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FOREWORD

This report presents the results of studies on the relationship between air-blast injury and impairment of pulmonary function in dogs and sheep. Specifically, impairment of pulmonary function was measured in terms of alterations in the venous-arterial shunt, ineffective alveolar ventilation, and blood gas tensions.

The findings may be of interest to those involved in the analysis of weapons effects or in industrial or military medicine.

This study is part of a broad program in the field of Blast and Shock Biology, the aims of which are the accurate prediction of hazards from explosions and the development of a sound basis for the prognosis and treatment of blast injuries.

ABSTRACT

Pulmonary function tests were conducted before and after exposure of animals to air blasts produced in shock tubes or by high explosives. Pressure-time measurements were made with piezoelectric pressure transducers during each air-blast exposure. Blood samples were obtained without anesthesia from an indwelling arterial catheter. The blood P_{O_2}, P_{CO_2}, and pH and the end-tidal and mixed expired CO_2, O_2, and N_2 gas concentrations were measured for subjects breathing air and oxygen. There were increases in the alveolar-arterial O_2 differences $(A\text{-}a)_{O_2}$, and venous admixture $(\dot{Q}s/\dot{Q})$ which generally correlated with the extent of blast-induced lung damage. Calculations indicated that most of the increase in $(A\text{-}a)_{O_2}$ for subjects breathing air could be attributed to the increase in $\dot{Q}s/\dot{Q}$ alone. The threshold for lung injury resulting in increased venous admixture in sheep was about 20 psi for reflected overpressures of "long" duration. Pressures above 43 psi usually caused severe lung damage in which the venous-arterial shunt exceeded 30 percent of the cardiac output, a condition in which the arterial oxygen tension was below the level required for full saturation of the hemoglobin even with animals breathing pure oxygen.

ACKNOWLEDGMENTS

The authors would like to acknowledge the able technical assistance of Messrs. David A. Byrne, Bill B. Daughtery, Allie Shaw, Charles S. Gaylord, and Glen L. Paxton in the Department of Comparative Environmental Biology and to express appreciation to Mmes. Treva J. Miller, Aida Elliott, and Wanda L. Deyo and others in the Department of Physiology who provided technical guidance and assistance in the analytical work.

Appreciation is expressed to Mrs. Maxine U. Thibert for editing and typing the manuscript and to Mr. Takeshi Minagawa for assistance with the illustrations.

We would also like to express appreciation to Messrs. Wilmer R. Kerzee and Bill Garrett, and Mrs. Sherry Schluter for programming and conducting the calculations on the Burroughs B5500 electronic computer.

Finally, we would like to express sincere appreciation to Ernest A. Henderson, D.V.M., for veterinary surgical support in the development and refinement of techniques of implanting the arterial catheters according to procedures initiated by R. W. Dougherty, D.V.M., USDA Animal Disease Control Center, Ames, Iowa.

The experimental work discussed in this manuscript was conducted according to the principles enunciated in the "Guide for Laboratory Animal Facilities and Care," prepared by the National Academy of Sciences-National Research Council.

TABLE OF CONTENTS

LIST OF FIGURES

THE ACUTE EFFECTS OF AIR BLAST ON PULMONARY FUNCTION IN DOGS AND SHEEP

Edward G. Damon, John T. Yelverton, Ulrich C. Luft,
Kabby Mitchell, Jr., and Robert K. Jones

INTRODUCTION

The lungs are more susceptible to primary blast injury than other vital organs. Consequently, considerable attention has been devoted to the documentation of the extent and nature of lung-blast injuries resulting from various levels and conditions of blast exposure.[1-9] To date, however, most of these have dealt only with the pathological alterations associated with air-blast damage. Studies of the physiological effects of blast on pulmonary function have been limited to investigation of the rate, depth, and rhythm of respiration;[1-3, 9-10] diaphragmatic action potentials;[10] and lung compliance, respiration, and gas exchange in rabbits.[11, 12]

The cardiopulmonary system exchanges and transports respiratory gases by means of two pumps: an air pump (the pulmonary system) and a fluid pump (the cardiovascular system). Normally, the outputs of these two pumps, the cardiac output ($\dot{Q}$) and the alveolar ventilation ($\dot{V}_A$), are coordinated in such a way as to result in an efficient system of gas exchange which maintains within narrow limits the pH, CO_2 content, and oxygen saturation of the blood. In lung-blast injury, the membranes separating the two systems are disrupted, resulting in hemorrhage into the air-containing spaces (alveoli, alveolar ducts, bronchioles, and bronchi) and may result in injection of air bubbles into the circulatory system.[1, 4, 8] Until adequate compensation occurs in the surviving organism, such injuries might be expected to produce, among other effects, a mismatching of the outputs of the two pumps; i.e., aberrations in the $\dot{V}_A/\dot{Q}$ ratios in

various parts of the lungs. One might expect that some level of circulation would be maintained through damaged, nonventilated parts of the lungs, resulting in an admixture of venous blood with the oxygenated blood and similarly that there would be ventilation of parts of the lungs in which the pulmonary circulation had been disrupted by blast injury which would be ineffective for gas exchange and would, therefore, constitute alveolar dead space ventilation. A fluid shift from capillaries to interstitial and alveolar spaces may also occur resulting in the development of pulmonary edema. In order to provide a sound basis for the intelligent prognosis and therapy of blast survivors and to provide data for prediction of the physiological effects of overpressures, there is a distinct need for an investigation designed to explore the relationship between the level of blast exposure and the resultant changes in functional efficiency of the cardiopulmonary system. Such information is also needed for proper interpretation of the effects of exposure to combinations of air blast and other environmental stresses.

Therefore, the following study was performed to investigate the acute effects of various levels of air-blast injury on pulmonary ventilation and gas exchange in sheep and dogs with major emphasis on changes produced in the venous-arterial shunt and attendant effects on the blood-gas parameters.

METHODS

General

The abbreviations and symbols used in the text are defined in Appendix A. The effects of air-blast injury on the venous-arterial shunt ($\dot{Q}s/\dot{Q}$), alveolar-arterial O_2 gradient $(A\text{-}a)_{O_2}$, arterial-alveolar (end-tidal) CO_2 gradient $(a\text{-}A)_{CO_2}$, alveolar dead space ventilation, oxygen tension ($P_{a_{O_2}}$), carbon dioxide tension ($P_{a_{CO_2}}$), and arterial blood pH

were investigated in sheep and dogs. Pulmonary function tests with and without anesthesia were conducted before and as soon as possible (usually within 30 minutes) after exposure to air blast. Each animal's pre-exposure test data served as its own controls.

Animals

Thirty-six young adult ewes and seven Beagles were utilized. The mean body weights were 42.2 and 8.2 kg for sheep and dogs, respectively. Twenty-seven of the sheep and six of the dogs were exposed to air blast. The rest of the animals were used as controls and to develop the pulmonary function test procedures.

Air-Blast Exposures

With the exception of three sheep exposed to blast from an air burst of a 64-lb spherical charge of TNT, all blast-response tests in this study were conducted in shock tubes. The ambient pressure at exposure was the local barometric pressure (12 psia).[13] The sheep were exposed with their left sides against the endplate of a 42-72 inch diameter shock tube to "sharp"-rising reflected overpressures (P_f) with durations ranging from 93 to 225 msec.[14, 15] The dogs were exposed with their left sides against the endplate of a 24-40 inch diameter shock tube to reflected overpressures of 335 to 380 msec duration.[16] Each animal was secured to the endplate by means of a harness constructed of nylon webbing.

Pressure-time measurements were made during each test with piezoelectric pressure transducers. The pressure-time instrumentation has been previously described.[14] Figure 1 presents a representative pressure-time waveform that is typical of those recorded by a gauge mounted side-on in the walls of the 6-ft shock tube directly above the back of the sheep.

Sampling of Arterial Blood from Anesthetized Animals

Dogs were anesthetized with an intravenous dose (25 mg/kg) of

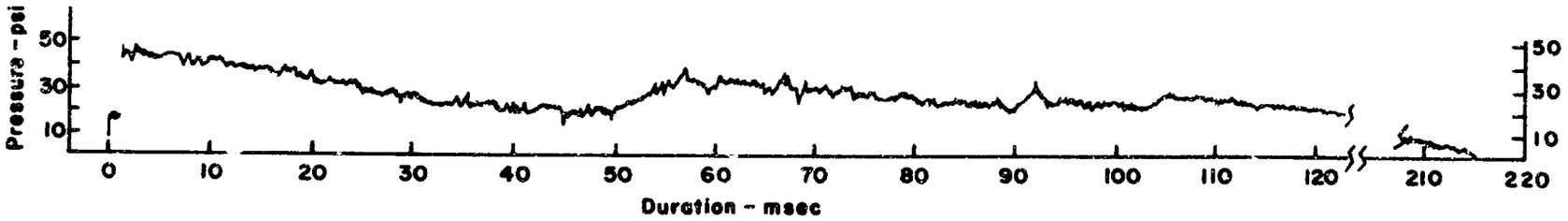


Figure 1. Typical pressure-time waveform recorded by a gauge mounted 6 inches upstream from the endplate of the 42-72-inch diameter shock tube.

sodium pentobarbital. Atropine sulfate (0.05 mg/kg) and sodium secobarbital (20 mg/kg) were used for anesthesia in sheep.

An 18 gauge cournand arterial needle was inserted into the surgically exposed femoral or carotid artery. A plastic three-way stopcock with a rubber cap on its main outlet was mounted on the cournand needle. The dead space of a 2 or 5 cc glass syringe fitted with a 20 gauge needle was filled with dilute heparin (10 mg/ml) and air-free blood samples were drawn by inserting the needle through the rubber cap on the stopcock. The syringe was capped with a Luer-Lok cap, placed in an ice bath and the blood usually analyzed within 1 to 5 minutes after sampling.

Sampling of Arterial Blood Without Anesthesia

Several techniques for sampling arterial blood without anesthesia were tried with varying degrees of success.[17, 18] The most successful procedure consisted of the insertion of a polyvinyl tube through the femoral artery into the caudal part of the aorta of the sheep (Fig. 2). This technique has been described in a separate report.[19] Blood was drawn from carotid arteries in dogs.

In some sheep, the indwelling cannula remained functional for more than 140 days after surgery. The location of the cannula on the back of the animal was convenient for obtaining the arterial blood samples at the desired times during the pulmonary function tests. Blood samples from the cannula were drawn and processed as for anesthetized animals.

Blood Gases and pH

The pH, $P_{a_{O_2}}$, and $P_{a_{CO_2}}$ of the blood were measured with either an Instrumentation Laboratories Model 113-SI Ultra-Micro pH, P_{O_2}, and P_{CO_2} Blood Analyzing System, or a Beckman Model 160 Physiological Gas Analyzer with Modular Cuvette and oxygen macroelectrode. All three measurements were made at a temperature of 37° C and then corrected to the species body temperature (39° C for both sheep and dogs).[20, 21] The

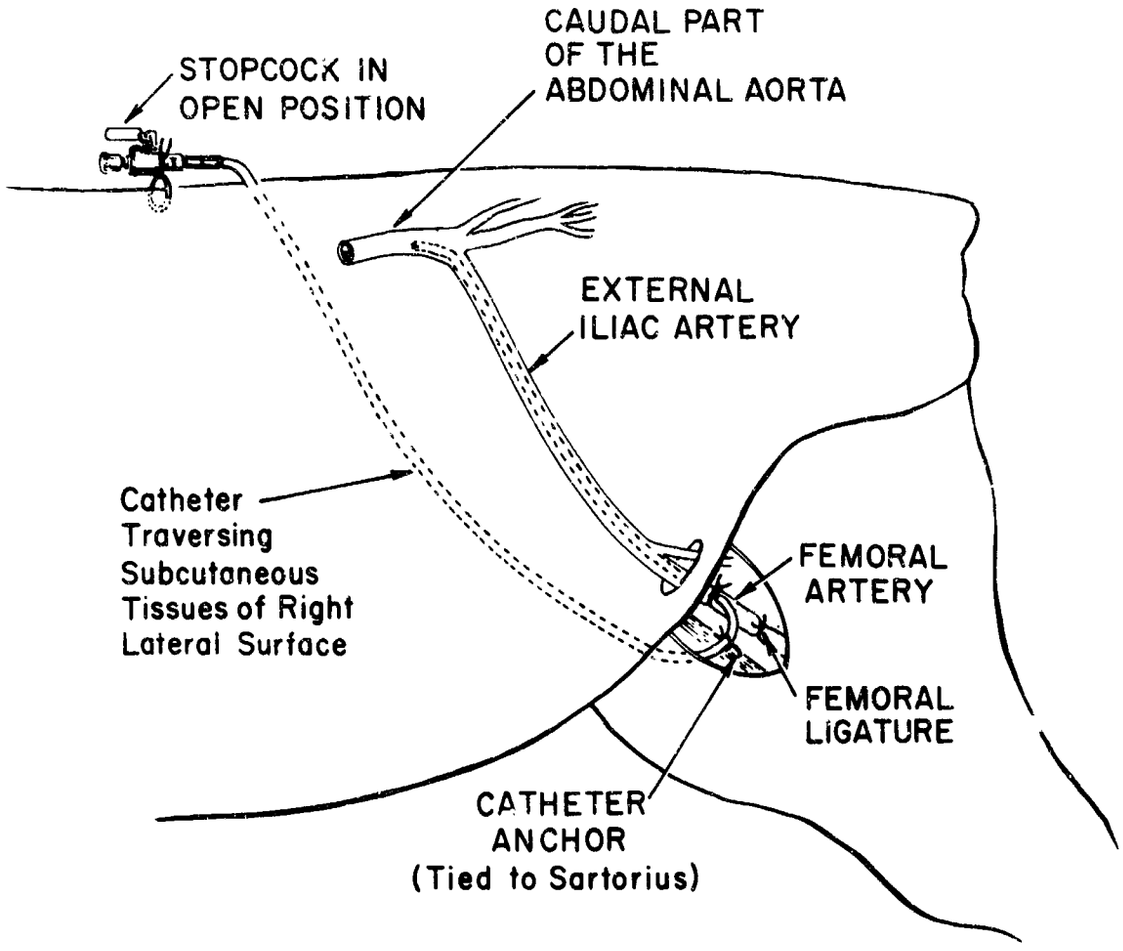


Figure 2. Cannulation of femoral artery for repeated sampling of arterial blood in unanesthetized sheep.[19]

electrodes were calibrated immediately before and after each analysis. All calibration gas mixtures were analyzed by the micro Scholander technique.[22]

Expired Gases

During the blood sampling process, inspired and expired carbon dioxide and nitrogen concentrations were monitored continuously with a Beckman Spinco Model LB-1 CO_2 Analyzer with linearizer and a Med Science Electronics Model 305 AR nitrogen analyzer, respectively. The respiratory traces from these instruments were recorded with a Honeywell Model 1508 Visicorder. The animals were connected directly to the breathe-through sample cell of the CO_2 analyzer by means of a latex mask (Fig. 3). A low dead space, Hans-Rudolph type, two-way breathing valve was connected by a short piece of tygon tubing to the back of the breathe-through sample cell. The mask and instrument dead space was approximately 90 cc. The mixed expired gases were collected in 30-liter Douglas bags and analyzed for O_2 and CO_2 concentration by both the micro Scholander technique and the blood-gas electrodes described above. Expired gas volumes were measured with a dry gas meter and corrected to the body temperature and pressure saturated with water vapor (BTPS). Oxygen consumption ($\dot{V}_{O_2}$) and CO_2 elimination ($\dot{V}_{CO_2}$) were corrected to standard temperature and pressure, dry (STPD).

CALCULATIONS

All calculations were performed on a Burroughs B5500 electronic computer.

Alveolar Gas Tensions and Pulmonary Ventilation

The $P_{A_{CO_2}}$ values were calculated from the mean end-tidal (E.T.) CO_2 concentrations which were recorded during the blood sampling with the animals breathing either room air or oxygen. The procedure for cal-

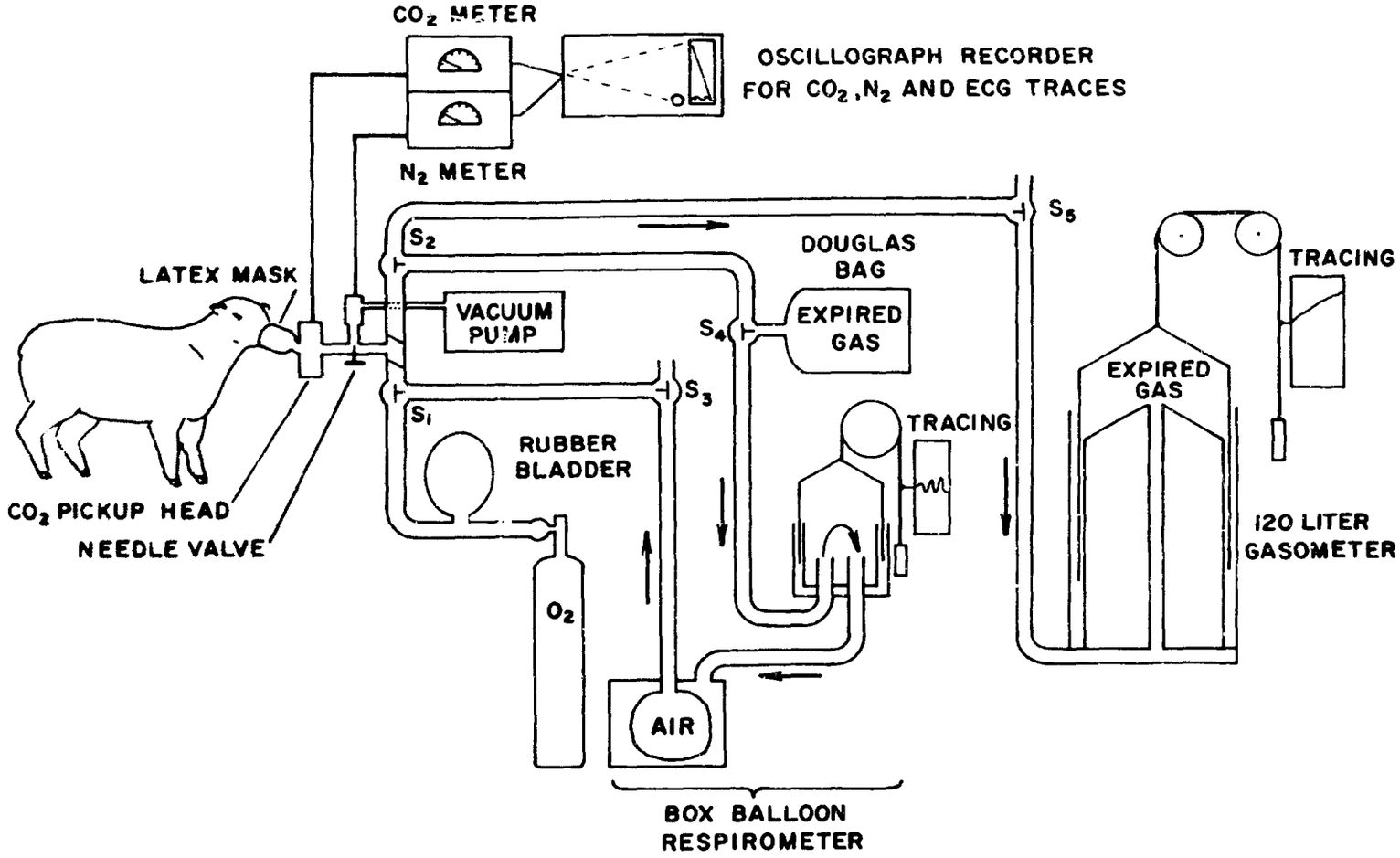


Figure 3. Schematic of equipment for conducting pulmonary function tests on unanesthetized sheep.

culating the alveolar gas tension, alveolar ventilation, and percent ineffective alveolar ventilation (alveolar dead space) were as described in Reference 23 and were corrected to the species body temperature (39° C).

In the room-air tests, the volume of inspired gas ($\dot{V}_I$) was calculated from the measured volume of expired gas ($\dot{V}_E$) and corrected to BTPS.[23]

Venous-Arterial Shunt

An oxygen method was used to measure venous-to-arterial shunt as a fraction of the cardiac output.[24] For animals breathing pure oxygen with the hemoglobin fully saturated, the following equation was used:

$$\dot{Q}s/\dot{Q} = \frac{0.0031\ (P_{A_{O_2}} - P_{a_{O_2}})}{0.0031\ (P_{A_{O_2}} - P_{a_{O_2}}) + (C_{a_{O_2}} - C_{\bar{v}_{O_2}})} \qquad (1)$$

where $\dot{Q}s/\dot{Q}$ = the venous-arterial shunt as a fraction of the cardiac output; $P_{A_{O_2}} - P_{a_{O_2}}$ = the difference in oxygen tension between the alveolar gas and the arterial blood; 0.0031 = the oxygen solubility factor for plasma; and $C_{a_{O_2}} - C_{\bar{v}_{O_2}}$ = the arteriovenous oxygen content difference $(a\text{-}\bar{v})_{O_2}$.

The $(a\text{-}\bar{v})_{O_2}$ difference remains fairly constant at resting levels of O_2 consumption and cardiac output. An average value for $(a\text{-}\bar{v})_{O_2}$ difference in these animals was obtained by cardiac catheterization of five sheep. The oxygen content of arterial blood and mixed venous blood was measured by the Van Slyke method. The following means and ranges of the $(a\text{-}\bar{v})_{O_2}$ differences were obtained:

	Number of Determinations	$(a-\bar{v})O_2$ Difference	
		Mean	Range
Nonanesthetized Animals Breathing Room Air	5	4.2	3.0-6.0
Nonanesthetized Animals Breathing O_2	2	5.5	4.1-6.9
Anesthetized Animals Breathing Room Air	2	5.3	4.3-6.3
	Mean	5.0	

The overall mean value of 5.0 was used for both sheep and dogs in all calculations involving $(a-\bar{v})O_2$ difference.

For animals with P_{aO_2} less than 120 mm Hg, the hemoglobin would not be 100 percent saturated and, therefore, equation (1) would not apply. In these cases, the percent saturation of the blood (S_{O_2}) was derived from the measured P_{aO_2} by using oxygen dissociation curves[25] and equation (1) modified as follows:

$$\dot{Q}s/\dot{Q} = \frac{O_2 \text{ Capacity } (1 - S_{O_2}) + 0.0031 (P_{AO_2} - P_{aO_2})}{O_2 \text{ Capacity } (1 - S_{O_2}) + 0.0031 (P_{AO_2} - P_{aO_2}) + 5.0} \qquad (2)$$

The mean and standard deviation of 12 pre-exposure determinations of O_2 capacity in sheep was 14.5 ± 1.71 ml O_2/100 ml blood. For sheep in which the O_2 capacity was not measured, this pre-exposure mean was used for calculation of $\dot{Q}s/\dot{Q}$. For dogs, the value used was 20.0 ml O_2/100 ml blood.

Breakdown of Causes of Alveolar-Arterial Oxygen Differences

$(A-a)O_2$ for Animals Breathing Air

The estimation of that portion of the $(A-a)O_2$ gradient due to venous admixture alone was based upon the following equation, the derivation of

which has been previously described:[26]

$$C_{a'O_2} = C_{c_{O_2}} - \dot{Q}s/\dot{Q} \frac{5.0}{1 - \dot{Q}s/\dot{Q}} \quad (1)$$

where $C_{a'O_2}$ = virtual $C_{a_{O_2}}$ due to shunt and 5.0 = $(a-\bar{v})_{O_2}$ difference.

The procedure was to obtain $S_{c_{O_2}}$ from the O_2 dissociation curve for the pH and $P_{A_{O_2}}$ of the subject and then calculate $C_{c_{O_2}}$ from:

$$C_{c_{O_2}} = S_{c_{O_2}} (O_2 \text{ capacity}) + 0.0031\ P_{A_{O_2}}. \quad (2)$$

$C_{a'O_2}$ was then calculated from (1) using $C_{c_{O_2}}$ and $\dot{Q}s/\dot{Q}$ as inputs. $S_{a'O_2}$ was obtained from $C_{a'O_2}$ and the O_2 capacity. $P_{a'O_2}$ was then read from the O_2 dissociation curve for $S_{a'O_2}$ and the pH.[25]

$P_{A_{O_2}} - P_{a'O_2}$ gave the part of the alveolar-arterial oxygen tension difference due solely to the venous-arterial shunt. The remainder of the $(A-a)_{O_2}$ difference was due to impairment of diffusion capacity or aberrations in the $\dot{V}_A/\dot{Q}$ ratio.[26,27]

RESULTS

The results of the study are illustrated in Figures 4 through 7 and presented in Tables 1 through 7 in the Appendix. The most significant findings are reviewed in the following sections.

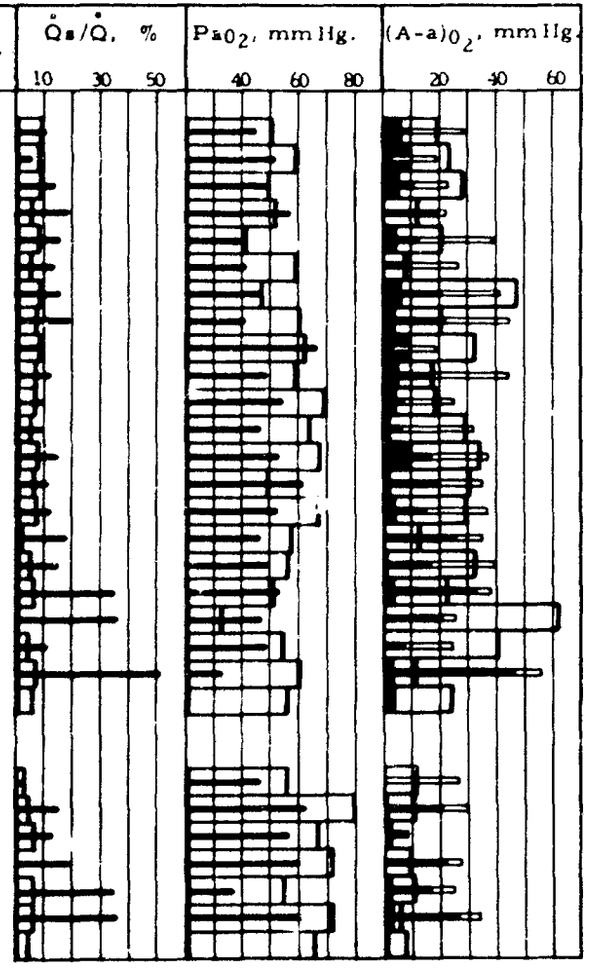
Venous-Arterial Shunt

A. Pre-Exposure Tests

Totals of 43 pre-shot determinations of the percent venous-arterial shunt ($\dot{Q}s/\dot{Q}$) in 25 unanesthetized sheep and 24 determinations of $\dot{Q}s/\dot{Q}$ in 19 anesthetized sheep were conducted. The mean values, standard deviations, and ranges were 7.2 ± 1.89 percent (3.5-10.6), and 7.8 ± 2.38 percent (3.5-11.2) for unanesthetized and anesthetized sheep, respectively. In

EFFECTS OF AIR BLAST ON VENOUS-ARTERIAL SHUNT ($\dot{Q}s/\dot{Q}$), ARTERIAL 0_2 TENSION (Pa_{0_2}), AND ALVEOLAR-ARTERIAL 0_2 GRADIENT $(A\text{-}a)_{0_2}$, IN SHEEP AND DOGS

Animal Number	Lung Weight Per Cent of Body Weight	Lung Hemorrhage	Reflected Pressure, psi
Sheep			
381[a]	0.96	None	10
401[a]	0.89	Petechial	17
372[a]	0.92	Petechial	21
386[a]	1.14	Small Isolated	21
366[a]	0.92	Small Isolated	32[b]
570			33
715			34
646			34
811			35
794			38
000			40
771			40
750			40
187	1.21	Confluent	41
172	1.17	Confluent	42
384[a]	1.31	Confluent	74[b]
821			42
181	2.33	Entire Lobes	43
487[a]	2.54	Entire Lobes	122[b]
825			45
407[a]	2.51	Entire Lobes	48
		Control mean	
Beagles			
D-56[a]	2.44	Confluent	36
D-19	1.47	Confluent	37
D-43	1.12	Confluent	40
D-22	3.85	Entire Lobes	39
D-44	2.65	Entire Lobes	39
D-53[a]	3.77	Entire Lobes	42
		Control mean	



a = Anesthetized
b = Exposed to 64-lb charge of TNT

☐ Pre-exposure
— Post-exposure

Note: Shaded part of $(A\text{-}a)0_2$ bar is portion due to shunt only.

Figure 4.

EFFECTS OF AIR BLAST ON VENOUS-ARTERIAL SHUNT ($\dot{Q}s/\dot{Q}$), ARTERIAL 0_2 TENSION (P_{a0_2}), AND ALVEOLAR-ARTERIAL 0_2 GRADIENT $(A\text{-}a)0_2$, IN SHEEP AND DOGS

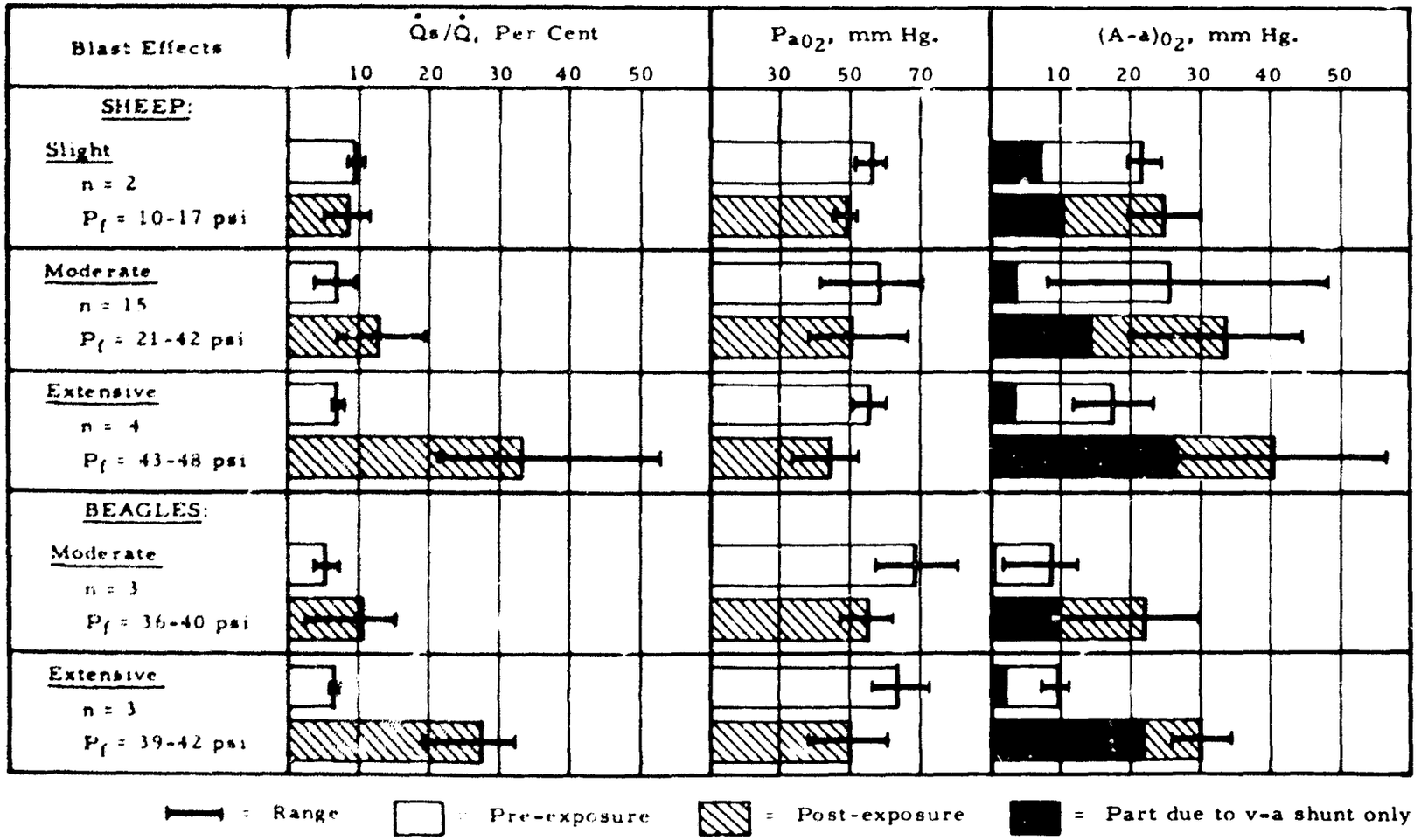


Figure 5.

EFFECTS OF AIR BLAST ON RESPIRATORY RATE, CO_2 TENSION (Pa_{CO_2}), AND pH OF THE ARTERIAL BLOOD FOR ANIMALS BREATHING AIR

Animal Number	Lung Weight, Per Cent of Body Weight	Lung Hemorrhage	Reflected Pressure, psi
Sheep			
381[a]	0.96	None	10
401[a]	0.89	Petechial	17
372[a]	0.92	Petechial	21
386[a]	1.14	Small Isolated	21
365[a]	0.92	Small Isolated	32[b]
570			33
715			34
646			34
811			35
794			38
000			40
771			40
750			40
187	1.21	Confluent	41
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825			45
407[a]	2.51	Entire Lobes	48
		Control mean	
Beagles			
D-56[a]	2.44	Confluent	36
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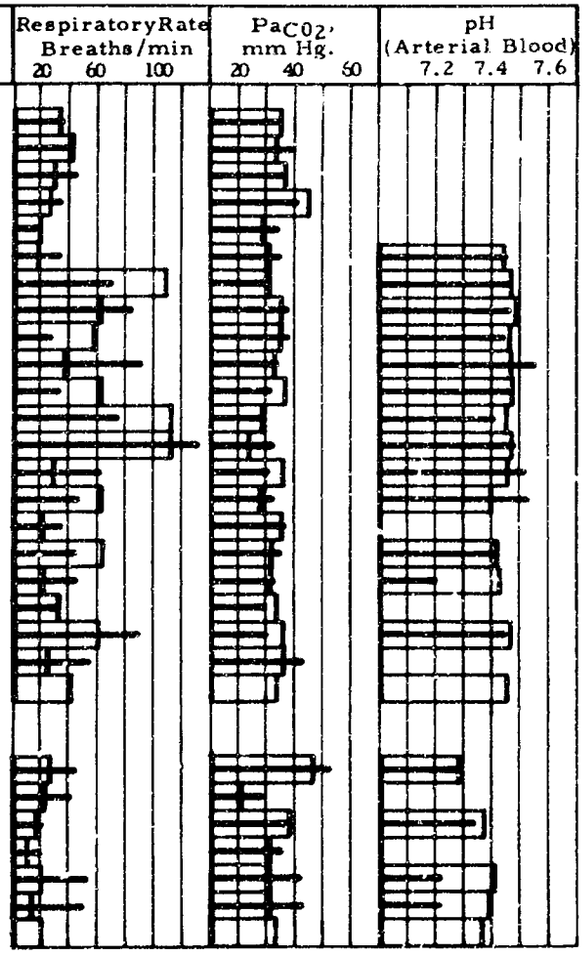
D-44	2.65	Entire Lobes	39
D-53[a]	3.77	Entire Lobes	42
		Control mean	



a = Anesthetized
b = Exposed to 64-lb charge of TNT

□ Pre-exposure
▬ Post-exposure

Figure 6.

EFFECTS OF AIR BLAST ON VENTILATION ($\dot{V}_I$), CO_2 GRADIENT $\left[(a\text{-}E.T.)_{CO_2}\right]$, AND INEFFECTIVE ALVEOLAR VENTILATION $\left[(a\text{-}E.T.)_{CO_2}/Pa_{CO_2}\right]$ IN ANIMALS BREATHING AIR

Animal Number	Lung Weight, Per Cent of Body Weight	Lung Hemorrhage	Reflected Pressure, psi
Sheep			
381[a]	0.96	None	10
401[a]	0.89	Petechial	17
372[a]	0.92	Petechial	21
386[a]	1.14	Small Isolated	21
366[a]	0.92	Small Isolated	32[b]
570			33
715			34
646			34
811			35
794			38
000			40
771			40
750			40
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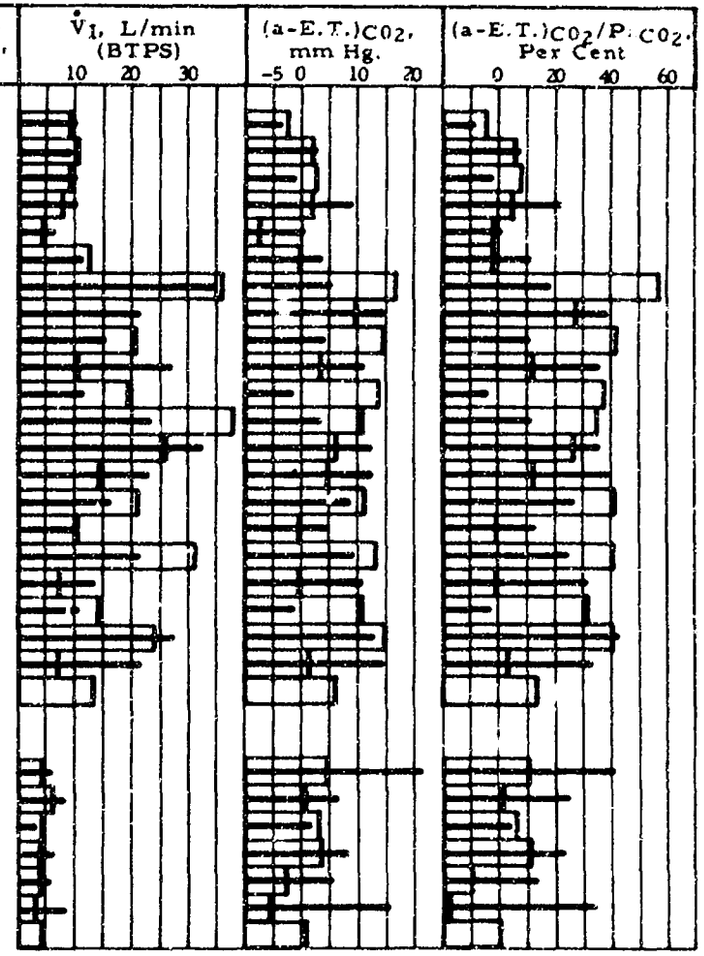
487[a]	2.54	Entire Lobes	122[b]
825			45
407[a]	2.51	Entire Lobes	48
		Control mean	
Beagles			
D-56[a]	2.44	Confluent	36
D-19	1.47	Confluent	37
D-43	1.12	Confluent	40
D-22	3.85	Entire Lobes	39
D-44	2.65	Entire Lobes	39
D-53[a]	3.77	Entire Lobes	42
		Control mean	



a = Anesthetized
b = Exposed to 64-lb charge of TNT

□ Pre-exposure
— Post-exposure

Figure 7.

10 of the above cases, tests with and without anesthesia were conducted on the same animals. The mean differences of the two tests for the 10 animals was 0.60 percent, which was not significant ($0.5 > P > 0.4$). The results of tests both with and without anesthesia are summarized in the following sections.

The mean, standard deviation, and range for five pre-exposure determinations of $\dot{Q}s/\dot{Q}$ in Beagles was 5.6 + 1.3 percent (3.8-6.9). Three of these tests were conducted without anesthesia.

B. Post-Exposure Tests

Post-exposure tests were completed on 21 sheep and 6 Beagles. Eleven of the sheep and all of the dogs were sacrificed immediately upon completion of the pulmonary function tests to assess the extent of lung-blast injury. The remainder of the animals were retained for studies of the chronic effects of air blast on pulmonary function, the results of which will be presented in a future report. Figure 4 lists the lung weights and extent of lung hemorrhage for the sacrificed animals, the reflected over-pressure to which each animal was exposed, and illustrates the pre- and post-exposure $\dot{Q}s/\dot{Q}$ for each. The animals for which no lung weight data are presented were retained for follow-up studies of the blast-injury recovery pattern. Also shown in Figure 4 are the $P_{a_{O_2}}$ and the alveolar-arterial O_2 differences for animals breathing air. The control mean values presented in this and subsequent figures are for all pre-exposure tests. The figure indicates that with but two exceptions (sheep No. 811 and Beagle No. D-56) all animals exposed to "long"-duration reflected pressures in the range of 20 to 40 psi exhibited variable increases in the post-exposure $\dot{Q}s/\dot{Q}$. The two sheep exposed to reflected pressures of 10 and 17 psi did not exhibit increased venous-arterial shunt. At the higher pressure levels, increases in the $\dot{Q}s/\dot{Q}$ were generally correlated with increasing levels of blast lung injury as indicated by the lung weight data and the extent of

lung hemorrhage observed at autopsy. This correlation was more evident in the dogs than in the sheep.

Concurrent with increases in $\dot{Q}s/\dot{Q}$, there were usually decreases in the arterial oxygen tensions ($P_{a_{O_2}}$) and increases in the alveolar-arterial oxygen gradients $(A\text{-}a)_{O_2}$ for animals breathing air (Fig. 4). As indicated by the shaded portions of the $(A\text{-}a)_{O_2}$ bars in Figure 4, most of the post-blast increases in $(A\text{-}a)_{O_2}$ in the dogs were due to a v-a shunt alone. For example, the mean post-exposure increase in the $(A\text{-}a)_{O_2}$ gradient in the six Beagles was 16.6 mm Hg, of which 14.2 mm Hg were due to shunt and 2.4 mm Hg to the other two factors that affect the $(A\text{-}a)_{O_2}$ gradient; namely, the diffusion capacity and aberrations in the $\dot{V}_A/\dot{Q}$ ratio.[26, 27] The portion of the $(A\text{-}a)_{O_2}$ gradient due to factors other than the shunt (represented by the clear portions of the bars in Fig. 4) changed very little after the blast. Although there were inconsistencies in these relationships in the sheep data, the pattern described above is clearly indicated by the mean values obtained when the animals were grouped according to level-of-blast injury as shown in Figure 5. The sheep were divided into blast-response groups designated as slight, moderate, or extensive on the basis of the degree of lung hemorrhage and/or overpressure. The dogs were similarly divided into two groups exhibiting moderate or extensive injuries. Figure 5 lists the number of animals, range of the reflected pressure (P_f), and shows the mean and range of the pre- and post-exposure $\dot{Q}s/\dot{Q}$, $P_{a_{O_2}}$, and $(A\text{-}a)_{O_2}$ for each group. The figure illustrates that, for sheep with no or only slight injuries, the mean, post-exposure, venous-arterial shunt was about the same as the pre-exposure value, whereas the arterial oxygen tension was slightly reduced and the $(A\text{-}a)_{O_2}$ gradient was slightly increased. For animals with moderate injuries, the mean post-exposure $\dot{Q}s/\dot{Q}$ was increased beyond the pre-exposure range, the $P_{a_{O_2}}$ was slightly reduced, and the $(A\text{-}a)_{O_2}$ was moderately increased

with most of the increase attributable to the change in $\mathring{Q}s/\mathring{Q}$. For animals with extensive injuries, there was a very marked increase in the mean post-exposure $\mathring{Q}s/\mathring{Q}$, a reduction in mean $P_{a_{O_2}}$ below the pre-exposure range and a marked increase in $(A\text{-}a)_{O_2}$, most of which was due solely to the increased venous-arterial shunt.

Respiratory Rate, CO_2 Tension, and pH of the Arterial Blood

The $P_{a_{CO_2}}$, pH, and respiratory rate data are summarized in Figure 6. In most cases, the post-exposure $P_{a_{CO_2}}$ values were within the normal range. There were slight increases in the post-exposure $P_{a_{CO_2}}$ values of all dogs except D-43, even though they exhibited increased respiratory rates. Dog D-56 had a slight pre-exposure respiratory acidosis ($P_{a_{CO_2}}$ = 47.0, pH = 7.29) that increased after air-blast exposure ($P_{a_{CO_2}}$ = 52.0, pH = 7.28). Sheep 181 showed metabolic acidosis which was only partially compensated by an increased respiratory rate ($P_{a_{CO_2}}$ = 33.0, pH = 7.20). Two of the most severely injured dogs had metabolic acidosis that was uncompensated by respiration even though their post-exposure respiratory rates were more than doubled ($P_{a_{CO_2}}$ = 42.0, pH = 7.22 and $P_{a_{CO_2}}$ = 43.0, pH = 7.23 for dogs D-44 and D-53, respectively).

Minute Volume, CO_2 Gradient, and Ineffective Alveolar Ventilation

The data in Figure 7 shows that the arterial, end-tidal, CO_2 difference $(a\text{-}E.T.)_{CO_2}$, and hence the percent ineffective alveolar ventilation, was generally correlated with the minute volumes in tests both with and without anesthesia. In addition to this, most of the dogs, and especially the two tested under anesthesia (D-56 and D-53), exhibited very marked post-exposure increases in the $(a\text{-}E.T.)_{CO_2}$ gradient which was attributable to increased alveolar dead space ventilation. In the sheep, changes in these parameters were variable and inconsistent.

Oxygen Consumption, Carbon Dioxide Elimination, and Respiratory Exchange Ratio

According to the data in Table 5 of the Appendix, the effects of air blast on oxygen consumption ($\dot{V}_{O_2}$), carbon dioxide elimination ($\dot{V}_{CO_2}$) and respiratory exchange ratio (R) were generally slight and inconsistent. Two of the more severely injured dogs (D-53 and D-44) exhibited slight decreases in both $\dot{V}_{O_2}$ and $\dot{V}_{CO_2}$ even though their pulmonary ventilation, following blast exposure, was increased (c.f., Tables 5 and 6). In one of the sheep (825) which was exposed to a reflected pressure of 45 psi and showed only a moderate increase in $\dot{Q}s/\dot{Q}$ (Fig. 4), there was a great increase in $\dot{V}_{O_2}$ and a reduction in $\dot{V}_{CO_2}$ so that R was reduced to 0.33. Consistent with the increased oxygen consumption was an elevation in the heart rate of this animal from a pre-shot value of 88 to 179 beats per minute after exposure.

DISCUSSION

The immediate post-exposure increase in the venous-arterial shunt, which was usually related to the extent of blast lung injury in these animals, indicates that there is a continuation of blood flow through blast-injured, nonventilated regions of the lungs resulting in an increase in the venous admixture. The increased venous admixture represents the composite effect of blood flow through regions of the lungs with atelectasis, intra-alveolar hemorrhage, airways blocked with hemorrhage, edema, or disruption of tissues by blast injury. This usually leads to hypoxia which persists even with increased ventilation.

Part of the increase in the venous-arterial shunt could also be due to pulmonary hypertension. Chronic pulmonary hypertension, due to causes other than trauma, has been reported to cause an elevation in the venous admixture attributed to an increase in the blood flow through anatomical arteriovenous shunts in the lungs.[28] That pulmonary hypertension

occurs in blast injury is indicated by the frequent occurrence of marked dilation of the right heart.[4]

It should be emphasized that in those animals that were exposed to "long"-duration overpressures at levels near or above the threshold for lethality ($\geq$43 psi), the lung injury was usually so severe that the v-a shunt exceeded 30 percent of the cardiac output and, in such cases, hypoxia was not entirely alleviated even with the subject breathing pure oxygen. One might conclude, therefore, that most of the survivors from exposure to blast overpressure at or above the lethal threshold level would probably be unable to perform tasks requiring exercise because of the lung injuries sustained. Furthermore, exercise, in such cases, may result in increased morbidity and mortality because of such attendant effects as: (1) increase in respiratory excursions of the lung with the possible consequence of further injection of air emboli into the circulation; (2) increase in cardiac output resulting in persistent pulmonary bleeding leading to a progressive increase in the venous-arterial shunt and reduction of $P_{a_{O_2}}$; and (3) progressive metabolic acidosis as a result of hypoxia and increasing anaerobic metabolism.

The fact that animals with lung injuries as slight as petechial hemorrhages showed an increase in the venous admixture indicates that the threshold pressures for lung injury from the standpoint of gross pathology ($\sim$20 psi for overpressures of "long" duration) may also be regarded as the threshold for impairment of pulmonary function. The main effect of slight lung injuries, however, would be a reduction in the pulmonary reserve which would cause respiratory distress only under conditions of severe exercise.

The pre-exposure mean CO_2 gradient for six dogs was 0.3 mm Hg with a range of -6 to 5 mm Hg (Table 3, Appendix B). After blast exposure, the mean value increased to 10 mm Hg with a range of 2 to 22 mm Hg.

The CO_2 gradient reflects the $\dot{V}_A/\dot{Q}$ ratio.[23] Negative CO_2 gradients occur during anesthesia or under conditions in which the ratio of the heart rate to the respiratory rate is high, as during heavy exercise.[29] Hence, the increases in the CO_2 gradients occurring in these dogs following air-blast exposure may have been partly the result of an increased respiratory rate relative to the heart rate, but may also have been due to lung-blast injury resulting in disruption of pulmonary circulation to alveoli in which ventilation was still occurring.[23] In any event, the result was an increase in the percent of the alveolar ventilation which was ineffective for gas exchange (Fig. 7). In most cases, these same effects were evident in the sheep data: the greatest post-exposure increases in the CO_2 gradient occurred in those animals with the highest venous-arterial shunt (c.f., Figs. 4 and 7). However, the changes in CO_2 gradients observed in these sheep were not as great as anticipated on the basis of results of earlier studies in which this parameter was measured on anesthetized sheep following exposure to explosive charges. In the latter case, 11 of 14 animals exhibited post-exposure CC_2 gradients above the rang of the pre-shot controls with values up to 29 mm Hg for animals with severe lung damage.[30]

The marked post-exposure reductions in the blood pH (Fig. 6) seen in two of the most severely injured dogs (D-44 and D-53) and one of the more severely injured sheep (181), coupled with CO_2 tensions in the normal or below normal range, indicate the occurrence of metabolic acidosis; probably resulting from a build-up of lactic acid due to hypoxia. Thus, it is the nature of severe lung-blast injury that increased ventilation may compensate or even overcompensate for a build-up in CO_2 in the blood but cannot fully compensate for the increased metabolic acidosis which results from hypoxia caused primarily by increased venous-arterial shunt.

The question arises as to the time required for recovery from lung-blast injury and the ability of the pulmonary system to compensate for such

injuries. The material presented in this report has been limited to the immediate effects of lung-blast injury on pulmonary function. The time-recovery patterns of the respiratory system from such injuries and the results of studies of the incidence of chronic, irreversible, or residual effects will be presented in a future report.

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APPENDIX A

ABBREVIATIONS AND SYMBOLS

Wherever possible, the standardized symbols and abbreviations recommended in Federation Proc. 9:602-605, 1950, are used:

General Symbols:		Examples:	
$\dot{X}$	= dot above any symbol indicates a time derivative	$\dot{V}$	= volume of gas, L/min
$\overline{X}$	= bar above any symbol indicates a mean value	$\overline{F}_{A_{CO_2}}$	= mean fractional concentration of carbon dioxide in alveolar gas

FOR GASES

Primary Symbols (large capital letters):		Examples:	
V	= gas volume	V_T	= tidal volume, ml
P	= gas pressure	$P_{A_{O_2}}$	= alveolar oxygen pressure, mm Hg
F	= fractional concentration in dry gas phase x 100, percent	$F_{I_{O_2}}$	= fractional concentration of oxygen in inspired gas
f	= respiratory frequency (breaths/min)		
R	= respiratory exchange ratio	R	= $\dot{V}_{CO_2}/\dot{V}_{O_2}$

Secondary Symbols (capital letter subscripts):		Examples:	
I	= inspired gas	$F_{I_{CO_2}}$	= fractional concentration of CO_2 in inspired gas
E	= expired gas	$\dot{V}_{E_{BTPS}}$	= volume of expired gas, body temperature and pressure, saturated, L/min

A = alveolar gas

E.T. = end-tidal gas

$\dot{V}_A$ = alveolar ventilation, L/min

$\dot{V}_D$ = anatomic dead space ventilation, L/min

P_B = barometric pressure

$(a\text{-}E.T.)_{CO_2}$ = arterial end-tidal CO_2 difference

STPD = standard temperature and pressure, dry (0° C, 760 mm Hg, dry)

BTPS = body temperature and pressure saturated with water vapor

ATPD = ambient temperature and pressure, dry

ATPS = ambient temperature and pressure saturated with water vapor

Primary Symbols (large capital letters):

Examples:

Q = volume of blood

$\dot{Q}$ = cardiac output, L/min

C = concentration of gas in blood phase

$C_{a_{O_2}}$ = ml O_2 in 100 ml arterial blood

S = percent saturation of Hb with O_2

$S_{a_{O_2}}$ = O_2 saturation of Hb of arterial blood

$\dot{Q}s/\dot{Q}$ = venous-arterial shunt = venous admixture with arterial blood in percent of cardiac output

$\dot{Q}s$ = blood flow through shunt

Secondary Symbols (small letters):

Examples:

a = arterial blood

$P_{a_{CO_2}}$ = partial pressure of CO_2 in arterial blood

v = venous blood

$C_{\bar{v}_{O_2}}$ = ml O_2 in 100 ml mixed venous blood

c = capillary blood

APPENDIX B - TABULATED DATA

Table 1 Effects of Air Blast on Arterial Oxygen ($P_{a_{O_2}}$) and CO_2 Tension ($P_{a_{CO_2}}$) for Dogs and Sheep Breathing Air or Oxygen

Table 2 Effects of Air Blast on Alveolar Oxygen ($P_{A_{O_2}}$) and CO_2 Tension ($P_{A_{CO_2}}$) for Dogs and Sheep Breathing Air or Oxygen

Table 3 Effects of Air Blast on Alveolar-Arterial Oxygen Difference $(A\text{-}a)_{O_2}$ and Arterial-End Tidal CO_2 Difference $(a\text{-}\bar{E}.T.)_{CO_2}$ for Dogs and Sheep Breathing Air or Oxygen

Table 4 Effects of Air Blast on Venous-Arterial Shunt ($\dot{Q}s/\dot{Q}$) and Blood pH for Dogs and Sheep

Table 5 Effects of Air Blast on CO_2 Elimination ($\dot{V}_{CO_2}$), Oxygen Consumption ($\dot{V}_{O_2}$), and Respiratory Exchange Ratio (R) for Dogs and Sheep Breathing Air

Table 6 Effects of Air Blast on Respiratory Rate (f), Inspiratory Minute Volume ($\dot{V}_I$), and Expiratory Minute Volume ($\dot{V}_E$) for Dogs and Sheep Breathing Air or Oxygen

Table 7 Effects of Air Blast on Total Alveolar Ventilation ($\dot{V}_A$) and Alveolar Dead Space Ventilation for Dogs and Sheep Breathing Air or Oxygen

TABLE 1

EFFECTS OF AIR BLAST ON ARTERIAL OXYGEN ($P_{a_{O_2}}$) AND CO_2 TENSION ($P_{a_{CO_2}}$) FOR DOGS AND SHEEP BREATHING AIR OR OXYGEN

Animal Number	Body Wt. Kg.	Reflected Pressure psi	Lung Weight, Per Cent of Body Weight	Lung Hemorrhage	$P_{a_{O_2}}$, mm Hg. (air) Pre[c]	(air) Post[c]	(oxygen) Pre[c]	(oxygen) Post[c]	$P_{a_{CO_2}}$, mm Hg. (air) Pre[c]	(air) Post[c]	(oxygen) Pre[c]	(oxygen) Post[c]
Sheep												
381[a]	39.5	10	0.96	None	53	46	354	328	36	36	20	28
401[a]	34.5	17	0.89	Petechial	60	52	398	468	34	40	26	27
372[a]	38.6	21	0.92	Petechial	49	50	370	264	37	36	48	48
386[a]	35.5	21	1.14	Small Isolated	53	58	425	152	45	41	53	52
366[a]	39.1	32[b]	0.92	Small Isolated	41	39	377	155	30	35	46	29
570	29.5	33	----		59	42	446	276	32	35	35	33
715	37.6	34	----		47	46	388	253	31	29	36	30
646	38.6	34	----		61	41	401	140	36	37	34	54
811	43.0	35	----		63	67	384	405	35	38	35	36
794	35.8	38	----		60	49	435	332	33	34	34	37
000	41.4	40	----		70	55	412	355	38	32	49	38
771	50.3	40	----		65	47	454	394	29	28	32	31
750	42.7	40	----		68	53	438	264	24	33	30	37
187	47.1	41	1.21	Confluent	49	62	440	358	36	31	38	33
172	42.3	42	1.17	Confluent	68	53	428	326	28	33	28	36
384[a]	35.5	74[b]	1.31	Confluent	58	47	421	167	36	37	49	46
821	38.5	42	----		58	50	459	274	32	36	39	40
181	47.1	43	2.33	Entire Lobes	51	53	437	82	33	33	34	34
487[a]	35.0	122[b]	2.54	Entire Lobes	34	48	--	74	34	30	--	33
825	40.8	45	----		55	48	465	353	36	31	37	36
407[a]	42.7	48	2.51	Entire Lobes	60	33	389	52	36	43	43	43
Beagles												
D-56[a]	8.6	36	2.44	Confluent	58	47	463	474	47	52	49	61
D-19	9.5	37	1.47	Confluent	81	63	452	260	22	28	25	32
D-43	9.5	40	1.12	Confluent	68	58	414	270	39	38	41	41
D-22	9.1	39	3.85	Entire Lobes	73	60	--	152	32	36	37	41
D-44	9.1	39	2.65	Entire Lobes	56	38	428	94	32	42	40	50
D-53[a]	4.8	42	3.77	Entire Lobes	62	51	422	92	32	43	34	51

a = Anesthetized
b = Exposed to 64-lb charge of TNT
c = Pre- and post-exposure

TABLE 2

EFFECTS OF AIR BLAST ON ALVEOLAR OXYGEN ($P_{A_{O_2}}$) AND CO_2 TENSION ($P_{A_{CO_2}}$) FOR DOGS AND SHEEP BREATHING AIR OR OXYGEN

Animal Number	Body Wt. Kg.	Reflected Pressure psi	Lung Weight, Per Cent of Body Weight	Lung Hemorrhage	$P_{A_{O_2}}$, mm Hg. (air) Pre[c]	(air) Post[c]	(oxygen) Pre[c]	(oxygen) Post[c]	$P_{A_{CO_2}}$, mm Hg. (air) Pre[c]	(air) Post[c]	(oxygen) Pre[c]	(oxygen) Post[c]
Sheep												
381[a]	39.5	10	0.96	None	72	75	553	543	38	40	19	26
401[a]	34.5	17	0.89	Petechial	85	72	546	552	31	37	23	18
372[a]	38.6	21	0.92	Petechial	78	73	538	525	34	37	32	43
386[a]	35.5	21	1.14	Small Isolated	65	80	523	519	42	32	47	52
366[a]	39.1	32[b]	0.92	Small Isolated	61	78	523	526	38	34	36	35
570	29.5	33	----		68	69	533	535	33	31	32	28
715	37.6	34	----		95	88	544	556	13	24	16	11
646	38.6	34	----		83	85	534	526	26	23	23	32
811	43.0	35	----		95	86	528	529	20	34	36	34
794	35.8	38	----		78	92	549	553	29	22	15	14
000	41.4	40	----		89	79	528	526	24	33	42	35
771	50.3	40	----		96	79	545	553	19	25	19	12
750	42.7	40	----		103	90	565	547	18	21	11	19
187	47.1	41	1.21	Confluent	79	97	540	557	32	19	29	15
172	42.3	42	1.17	Confluent	98	90	553	549	17	24	14	18
384[a]	35.5	74[b]	1.31	Confluent	72	82	479	512	37	32	46	38
821	38.5	42	----		91	90	557	551	19	27	19	20
181	47.1	43	2.33	Entire Lobes	74	91	547	556	33	23	24	12
487[a]	35.0	122[b]	2.54	Entire Lobes	95	74	---	509	23	31	--	27
825	40.8	45	----		97	74	557	553	21	17	14	16
407[a]	42.7	48	2.51	Entire Lobes	72	90	527	541	35	29	30	26
Beagles												
D-56[a]	8.6	36	2.44	Confluent	70	75	527	516	42	31	37	50
D-19	9.5	37	1.47	Confluent	92	92	545	546	21	21	24	24
D-43	9.5	40	1.12	Confluent	70	66	534	529	36	36	38	40
D-22	9.1	39	3.85	Entire Lobes	83	88	542	530	28	27	27	32
D-44	9.1	39	2.65	Entire Lobes	67	64	530	530	35	37	41	38
D-53[a]	4.8	42	3.77	Entire Lobes	70	85	535	543	38	28	34	26

a = Anesthetized
b = Exposed to 64-lb charge of TNT
c = Pre- and post-exposure

TABLE 3

EFFECTS OF AIR BLAST ON ALVEOLAR-ARTERIAL OXYGEN DIFFERENCE $(A\text{-}a)_{O_2}$ AND ARTERIAL-END TIDAL CO_2 DIFFERENCE $(a\text{-}E.T.)_{CO_2}$ FOR DOGS AND SHEEP BREATHING AIR OR OXYGEN

Animal Number	Body Wt. Kg.	Reflected Pressure psi	Lung Weight, Per Cent of Body Weight	Lung Hemorrhage	$(A\text{-}a)_{O_2}$, mm Hg. (air) Pre[c]	(air) Post[c]	(oxygen) Pre[c]	(oxygen) Post[c]	$(a\text{-}E.T.)_{CO_2}$, mm Hg. (air) Pre[c]	(air) Post[c]	(oxygen) Pre[c]	(oxygen) Post[c]
Sheep												
381[a]	39 5	10	0.96	None	20	30	199	215	-2	-3	1	2
401[a]	34.5	17	0.89	Petechial	24	20	148	84	2	3	3	3
372[a]	38.6	21	0.92	Petechial	29	23	168	261	3	-1	16	5
386[a]	35.5	21	1.14	Small Isolated	12	22	98	367	2	9	6	1
366[a]	39.1	32[b]	0.92	Small Isolated	20	39	198	279	-8	0	0	-7
570	29.5	33	----		8	26	87	259	-1	4	2	5
715	37.6	34	----		48	42	156	303	17	6	20	19
646	38.6	34	----		22	44	133	386	10	15	11	22
811	43.0	35	----		32	19	144	124	15	4	-2	1
794	35.8	38	----		18	44	114	221	4	12	19	23
000	41.4	40	----		18	24	116	171	14	-1	7	4
771	50.3	40	----		30	32	91	159	10	3	13	19
750	42.7	40	----		35	37	127	283	6	12	18	19
187	47.2	41	1.21	Confluent	30	34	100	199	4	12	9	18
172	42.3	42	1.17	Confluent	30	38	163	223	11	9	14	18
384[a]	35.5	74[b]	1.31	Confluent	13	34	58	345	-1	5	3	8
821	38.5	42	----		33	40	98	277	13	9	20	20
181	47.1	43	2.33	Entire Lobes	23	38	110	474	-1	10	10	21
487[a]	35.0	122[b]	2.54	Entire Lobes	61	26	---	433	11	-1	--	6
825	40.8	45	----		42	26	92	200	15	13	22	20
407[a]	42.7	48	2.51	Entire Lobes	12	56	138	490	1	14	12	17
Beagles												
D-56[a]	8.6	36	2.44	Confluent	12	28	64	42	5	22	13	10
D-19	9.5	37	1.47	Confluent	11	30	93	286	0	7	1	8
D-43	9.5	40	1.12	Confluent	2	9	120	259	2	2	3	1
D-22	9.1	39	3.85	Entire Lobes	11	28	---	377	4	8	9	8
D-44	9.1	39	2.65	Entire Lobes	11	26	102	436	-3	6	-2	11
D-53[a]	4.8	42	3.77	Entire Lobes	8	34	113	451	-6	15	-1	25

a = Anesthetized
b = Exposed to 64-lb charge of TNT
c = Pre- and post-exposure

TABLE 4

EFFECTS OF AIR BLAST ON VENOUS-ARTERIAL SHUNT ($\dot{Q}_s/\dot{Q}$)

AND BLOOD pH FOR DOGS AND SHEEP

Animal Number	Body Wt. Kg.	Reflected Pressure psi	Lung Weight, Per Cent of Body Weight	Lung Hemorrhage	$\dot{Q}_s/\dot{Q}$, %		pH, Arterial (air)		pH, Arterial (oxygen)	
					Pre[c]	Post[c]	Pre[c]	Post[c]	Pre[c]	Post[c]
Sheep										
381[a]	39.5	10	0.96	None	11.0	11.7	----	----	----	----
401[a]	34.5	17	0.89	Petechial	8.4	5.0	----	----	----	----
372[a]	38.6	21	0.92	Petechial	9.4	13.9	----	----	----	----
386[a]	35.5	21	1.14	Small Isolated	5.7	18.5	----	----	----	----
366[a]	39.1	32[b]	0.92	Small Isolated	8.3	15.4	----	----	----	----
570	29.5	33	----		5.1	13.8	7.44	7.45	7.40	7.41
715	37.6	34	----		8.8	15.8	7.47	7.46	7.47	7.40
646	38.6	34	----		7.6	19.3	7.49	7.47	7.53	7.38
811	43.0	35	----		8.2	7.2	7.47	7.45	7.33	7.38
794	35.8	38	----		6.6	12.1	7.47	7.55	7.46	7.52
000	41.4	40	----		6.7	9.6	7.48	7.46	7.29	7.44
771	50.3	40	----		5.4	9.0	7.45	7.41	7.47	7.37
750	42.7	40	----		7.3	14.9	7.47	7.49	7.47	7.47
187	47.1	41	1.21	Confluent	5.5	11.0	7.46	7.52	7.42	7.50
172	42.3	42	1.17	Confluent	6.5	12.1	7.41	7.54	7.42	7.50
384[a]	35.5	74[b]	1.31	Confluent	3.5	17.6	----	----	----	----
821	38.5	42	----		5.7	14.6	7.43	7.43	7.42	7.41
181	47.1	43	2.33	Entire Lobes	6.5	34.5	7.43	7.20	7.48	7.18
487[a]	35.0	122[b]	2.54	Entire Lobes	----	34.8	----	----	----	----
825	40.8	45	----		5.4	11.1	7.47	7.46	7.45	7.43
407[a]	42.7	48	2.51	Entire Lobes	7.9	53.4	----	----	----	----
Beagles										
D-56[a]	8.6	36	2.44	Confluent	3.8	2.6	7.29	7.28	7.28	7.18
D-19	9.5	37	1.47	Confluent	5.5	15.1	----	----	----	----
D-43	9.5	40	1.12	Confluent	6.9	13.8	7.37	7.35	7.35	7.24
D-22	9.1	39	3.85	Entire Lobes	----	19.0	----	----	----	----
D-44	9.1	39	2.65	Entire Lobes	5.9	32.0	7.42	7.22	7.35	7.13
D-53[a]	4.8	42	3.77	Entire Lobes	6.5	31.5	7.39	7.23	7.39	7.17

a = Anesthetized

b = Exposed to 64-lb charge of TNT

c = Pre- and post-exposure

TABLE 5

EFFECTS OF AIR BLAST ON CO_2 ELIMINATION ($\dot{V}_{CO_2}$), OXYGEN CONSUMPTION ($\dot{V}_{O_2}$) AND RESPIRATORY EXCHANGE RATIO (R) FOR DOGS AND SHEEP BREATHING AIR

Animal Number	Body Wt. Kg.	Reflected Pressure psi	Lung Weight, Per Cent of Body Weight	Lung Hemorrhage	$\dot{V}_{CO_2}$, ml/min STPD		$\dot{V}_{O_2}$, ml/min STPD		R	
					Pre[c]	Post[c]	Pre[c]	Post[c]	Pre[c]	Post[c]
Sheep										
381[a]	39.5	10	0.96	None	151	146	199	167	0.76	0.88
401[a]	34.5	17	0.89	Petechial	134	109	153	150	0.88	0.73
372[a]	38.6	21	0.92	Petechial	109	139	143	188	0.76	0.74
386[a]	35.5	21	1.14	Small Isolated	117	100	161	135	0.73	0.74
366[a]	39.1	32[b]	0.92	Small Isolated	78	139	131	179	0.60	0.78
570	29.5	33	----		154	137	264	238	0.59	0.57
715	37.6	34	----		135	294	258	411	0.52	0.72
646	38.6	34	----		177	194	254	322	0.70	0.60
811	43.0	35	----		172	278	211	275	0.82	1.01
794	35.8	38	----		103	226	158	291	0.65	0.78
000	41.4	40	----		151	138	214	175	0.70	0.79
771	50.3	40	----		283	203	371	357	0.76	0.57
750	42.7	40	----		213	233	227	336	0.94	0.69
187	47.1	41	1.21	Confluent	177	178	243	230	0.73	0.77
172	42.3	42	1.17	Confluent	146	160	198	202	0.75	0.79
384[a]	35.5	74[b]	1.31	Confluent	183	166	256	209	0.71	0.79
821	38.5	42	----		195	188	336	223	0.58	0.85
181	47.1	43	2.33	Entire Lobes	96	120	144	162	0.67	0.74
487[a]	35.0	122[b]	2.54	Entire Lobes	240	160	261	260	0.92	0.62
825	40.8	45	----		230	130	252	396	0.91	0.33
407[a]	42.7	48	2.51	Entire Lobes	82	198	119	209	0.69	0.95
Beagles										
D-56[a]	8.6	36	2.44	Confluent	34	30	42	49	0.83	0.62
D-19	9.5	37	1.47	Confluent	45	54	61	73	0.74	0.74
D-43	9.5	40	1.12	Confluent	60	44	90	70	0.67	0.63
D-22	9.1	39	3.85	Entire Lobes	49	61	67	73	0.73	0.84
D-44	9.1	39	2.65	Entire Lobes	50	39	82	65	0.62	0.60
D-53[a]	4.8	42	3.77	Entire Lobes	31	18	43	23	0.71	0.78

a = Anesthetized
b = Exposed to 64-lb charge of TNT
c = Pre- and post-exposure

TABLE 6

EFFECTS OF AIR BLAST ON RESPIRATORY RATE (f), INSPIRATORY MINUTE VOLUME ($\dot{V}_I$), AND EXPIRATORY MINUTE VOLUME ($\dot{V}_E$) FOR DOGS AND SHEEP BREATHING AIR OR OXYGEN

Animal Number	Body Wt. Kg.	Reflected Pressure psi	Lung Weight, Per Cent of Body Weight	Lung Hemorrhage	f, Breaths/min (air) Pre[c]	f, Breaths/min (air) Post[c]	f, Breaths/min (oxygen) Pre[c]	f, Breaths/min (oxygen) Post[c]	$\dot{V}_I$, L/min BTPS (air) Pre[c]	$\dot{V}_I$, L/min BTPS (air) Post[c]	$\dot{V}_E$, L/min BTPS (oxygen) Pre[c]	$\dot{V}_E$, L/min BTPS (oxygen) Post[c]
Sheep												
381[a]	39.5	10	0.96	None	36	38	17	11	9.54	10.22	15.33	9.58
401[a]	34.5	17	0.89	Petechial	43	43	13	14	10.97	9.47	11.88	6.93
372[a]	38.6	21	0.92	Petechial	31	47	17	34	9.29	10.33	7.69	9.12
386[a]	35.5	21	1.14	Small Isolated	27	36	25	53	8.03	10.24	7.60	--
366[a]	39.1	32[b]	0.92	Small Isolated	21	21	18	33	4.79	6.49	9.99	9.88
570	29.5	33	----		20	35	21	30	12.70	11.55	15.79	12.00
715	37.6	34	----		110	70	97	132	36.10	34.50	31.5	32.98
646	38.6	34	----		64	85	57	49	19.46	21.75	17.33	18.91
811	43.0	35	----		60	28	27	67	20.30	15.12	9.43	21.73
794	35.8	38	----		39	92	100	92	10.81	27.30	23.40	28.18
000	41.7	40	----		64	34	57	47	19.56	11.76	24.93	15.43
771	50.3	40	----		114	76	89	102	38.16	23.84	24.91	22.18
750	42.7	40	----		114	133	146	151	26.26	32.27	35.44	34.99
187	47.1	41	1.21	Confluent	29	62	38	78	14.41	23.28	18.43	30.76
172	42.3	42	1.17	Confluent	63	48	84	65	21.37	16.62	37.80	18.78
384[a]	35.5	74[b]	1.31	Confluent	23	36	14	32	10.47	10.44	6.90	8.46
821	38.5	42	----		66	46	71	39	31.54	21.84	6.66	22.31
181	47.1	43	2.33	Entire Lobes	22	46	42	50	7.28	13.54	7.18	17.90
487[a]	35.0	122[b]	2.54	Entire Lobes	34	31	--	34	13.96	10.89	---	9.13
825	40.8	45	----		62	90	96	98	24.38	27.90	22.14	20.82
407[a]	42.7	48	2.51	Entire Lobes	26	56	21	59	7.64	21.94	4.77	21.49
Beagles												
D-56[a]	8.6	36	2.44	Confluent	28	45	15	27	4.13	6.10	3.75	4.06
D-19	9.5	37	1.47	Confluent	24	42	20	29	6.18	8.22	4.71	9.70
D-43	9.5	40	1.12	Confluent	18	21	16	24	4.83	3.77	3.60	4.51
D-22	9.1	39	3.85	Entire Lobes	12	20	11	17	4.19	6.01	3.65	4.92
D-44	9.1	39	2.65	Entire Lobes	22	54	23	59	4.24	5.78	5.94	7.26
D-53[a]	4.8	42	3.77	Entire Lobes	14	50	11	48	3.52	8.40	3.31	--

a = Anesthetized
b = Exposed to 64-lb charge of TNT
c = Pre- and post-exposure

TABLE 7

EFFECTS OF AIR BLAST ON TOTAL ALVEOLAR VENTILATION ($\dot{V}_A$) AND ALVEOLAR DEAD SPACE VENTILATION FOR DOGS AND SHEEP BREATHING AIR OR OXYGEN

Animal Number	Body Wt. Kg.	Reflected Pressure psi	Lung Weight, Per Cent of Body Weight	Lung Hemorrhage	$\dot{V}_A$, L/min, BTPS (air) Pre[c]	$\dot{V}_A$, L/min, BTPS (air) Post[c]	$\dot{V}_A$, L/min, BTPS (oxygen) Pre[c]	$\dot{V}_A$, L/min, BTPS (oxygen) Post[c]	Alveolar Dead Space, % (air) Pre[c]	Alveolar Dead Space, % (air) Post[c]	Alveolar Dead Space, % (oxygen) Pre[c]	Alveolar Dead Space, % (oxygen) Post[c]
Sheep												
381[a]	39.5	10	0.96	None	3.44	3.21	10.28	5.65	-5.6	-9.0	6.3	6.8
401[a]	34.5	17	0.89	Petechial	3.69	2.57	7.08	3.79	6.5	8.0	12.1	15.0
372[a]	38.6	21	0.92	Petechial	2.78	3.30	1.41	3.04	8.0	-2.0	33.0	10.0
386[a]	35.5	21	1.14	Small Isolated	2.40	2.73	2.26	--	5.3	22.1	10.4	--
366[a]	39.1	32[b]	0.92	Small Isolated	1.79	3.53	2.04	3.98	-28.0	0.9	-0.7	-5.3
570	29.5	33	----		4.07	3.82	5.97	4.31	-3.3	10.4	6.8	14.4
715	37.6	34	----		8.73	10.75	7.90	13.75	56.5	18.9	54.8	62.4
646	38.6	34	----		5.98	7.49	4.99	8.79	27.7	39.5	32.6	41.4
811	43.0	35	----		7.41	7.16	3.57	6.86	42.0	11.1	-4.4	3.4
794	35.8	38	----		3.13	8.95	8.56	16.24	12.1	36.0	55.4	61.1
000	41.4	40	----		5.52	3.66	8.20	4.81	37.2	-3.8	13.8	9.0
771	50.3	40	----		12.90	7.03	7.88	9.16	35.1	11.6	41.2	60.7
750	42.7	40	----		10.48	9.52	20.77	11.49	26.6	36.2	62.5	49.7
187	47.1	41	1.21	Confluent	4.84	8.08	3.73	8.65	12.4	39.0	22.6	53.6
172	42.3	42	1.17	Confluent	7.75	5.71	16.00	6.12	40.7	26.7	49.8	49.6
384[a]	35.5	74[b]	1.31	Confluent	4.34	4.59	2.12	3.50	-1.6	13.7	5.9	18.3
821	38.5	42	----		8.95	6.13	1.85	7.59	41.3	25.4	51.5	49.5
181	47.1	43	2.33	Entire Lobes	2.50	4.62	2.30	7.66	-2.5	31.9	29.8	62.7
487[a]	35.0	122[b]	2.54	Entire Lobes	9.00	4.50	--	2.93	31.9	-3.7	--	18.5
825	40.8	45	----		9.31	6.48	7.90	7.73	40.6	43.0	60.4	54.6
407[a]	42.7	48	2.51	Entire Lobes	2.03	6.03	1.03	6.44	3.0	33.0	26.0	39.0
Beagles												
D-56[a]	8.6	36	2.44	Confluent	0.71	0.86	0.59	0.39	10.6	41.3	25.4	17.0
D-19	9.5	37	1.47	Confluent	1.84	2.19	1.48	1.46	1.9	24.4	2.9	26.7
D-43	9.5	40	1.12	Confluent	1.44	1.06	0.55	1.28	6.6	3.9	7.6	1.6
D-22	9.1	39	3.85	Entire Lobes	1.52	1.96	1.51	1.38	11.1	23.9	25.0	20.1
D-44	9.1	39	2.65	Entire Lobes	1.24	0.92	1.04	1.06	-10.2	13.3	-3.9	23.0
D-53[a]	4.8	42	3.77	Entire Lobes	0.71	0.65	0.85	--	-19.2	34.9	-1.8	--

a = Anesthetized
b = Exposed to 64-lb charge of TNT
c = Pre- and post-exposure

Unclassified

Security Classification

DOCUMENT CONTROL DATA - R & D

(Security classification of title, body of abstract and indexing annotation must be entered when the overall report is classified)

1. ORIGINATING ACTIVITY (Corporate author)
Lovelace Foundation for Medical Education & Research
Albuquerque, New Mexico

2a. REPORT SECURITY CLASSIFICATION
UNCLASSIFIED

2b. GROUP

3. REPORT TITLE
The Acute Effects of Air Blast on Pulmonary Function in Dogs and Sheep

4. DESCRIPTIVE NOTES (Type of report and inclusive dates)

5. AUTHOR(S) (First name, middle initial, last name)
Edward G. Damon; John T. Yelverton; Ulrich C. Luft; Kabby Mitchell, Jr.; and Robert K. Jones

6. REPORT DATE	7a. TOTAL NO. OF PAGES	7b. NO. OF REFS
March 1970	36	30

8a. CONTRACT OR GRANT NO.	9a. ORIGINATOR'S REPORT NUMBER(S)
DA-49-146-XZ-372	
b. PROJECT NO. NWER XAXM	
c. Task and Subtask A012	9b. OTHER REPORT NO(S) (Any other numbers that may be assigned this report)
d. Work Unit 01	DASA 2461

10. DISTRIBUTION STATEMENT
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11. SUPPLEMENTARY NOTES

12. SPONSORING MILITARY ACTIVITY
Director
Defense Atomic Support Agency
Washington, D. C. 20305

13. ABSTRACT

Pulmonary function tests were conducted before and after exposure of animals to air blasts produced in shock tubes or by high explosives. Pressure-time measurements were made with piezoelectric pressure transducers during each air-blast exposure. Blood samples were obtained without anesthesia from an indwelling arterial catheter. The blood P_{O_2}, P_{CO_2}, and pH and the end-tidal and mixed expired CO_2, O_2, and N_2 gas concentrations were measured for subjects breathing air and oxygen. There were increases in the alveolar-arterial O_2 difference $(A\text{-}a)O_2$, and venous admixture $(\dot{Q}s/\dot{Q})$ which generally correlated with the extent of blast-induced lung damage. Calculations indicated that most of the increase in $(A\text{-}a)_{O_2}$ for subjects breathing air could be attributed to the increase in $\dot{Q}s$ $\dot{Q}$ alone. The threshold for lung injury resulting in increased venous admixture in sheep was about 20 psi for reflected overpressures of "long" duration. Pressures above 43 psi usually caused severe lung damage in which the venous-arterial shunt exceeded 30 percent of the cardiac output, a condition in which the arterial oxygen tension was below the level required for full saturation of the hemoglobin even with animals breathing pure oxygen.

DD FORM 1 NOV 68 1473 REPLACES DD FORM 1473, 1 JAN 64, WHICH IS OBSOLETE FOR ARMY USE.

Unclassified

Security Classification

Security Classification

14. KEY WORDS	LINK A		LINK B		LINK C	
	ROLE	WT	ROLE	WT	ROLE	WT
Blast Injury						
Physiological Effects						
Shock						
Blood Gas Analysis						
Pulmonary Function						
Lung Trauma						
pH, P_{O_2}, P_{CO_2} in Sheep						
Dogs						
Overpressure						
Venous Admixture						
Alveolar Gas Tension						

Unclassified

Security Classification